ACUTE PERICARDITIS.

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This paper is based upon a study of the protocols of eighty proved cases of acute pericarditis and the literature particularly of the last five years. It seeks to emphasize certain physical signs which have seemed of importance to the writer.

Most authors, especially the writers of text-books, begin with a history of pericardial lesions. There is no record that Hippocrates observed the condition, but Galen noted it in animals and suspected it in man; various writers mentioned it, but it was not referred to as a clinical condition until described by Senac in 1749. Laennec, the father of auscultation, described a creaking sound over the heart, but it remained for Collin, his chief assistant, to clearly demonstrate the pericardial friction rub and interpret its clinical significance.

Anatomy. It is important for the student of pericardial inflammations to rehearse the anatomy of the pericardium—to have the anatomical relations of the sac to other structures in the thorax in mind is helpful in considering the subjective and objective symptomatology. The pericardium is a conical serofibrous sac enveloping the heart and the first portion of the great vessels. The apex is directed upward and surrounds the great vessels about two inches above their origin from the base of the heart. Its base is attached to the central tendon, and part of the adjoining muscular structure of the diaphragm extends a little farther to the left than to the right side. It is separated from the sternum in front by the remains of the thymus gland above and a little loose areolar tissue below, and is covered by the margins of the lungs, especially the left. Posteriorly it forms the anterior boundary of the posterior mediastinum resting upon the descending aorta, the bronchi, esophagus, trachea, and root of the left lung. Laterally it is covered by the pleurae, the phrenic nerve, with its accompanying vessels, descending between the two membranes on either side. There are two layers, a strong dense fibrous layer and the serous layer, which invests the surface of the heart—Cohnheim[1] observed that normally the parietal layer is always in immediate contact with the visceral.

Considering the intimate contact of the pericardium with almost all the structures of the thorax, it is not remarkable that pericarditis may be manifested in almost any portion of the thoracic viscera or even the abdominal, but still more remarkable that it may exist without giving a sign—Cohnheim pointed out that complete oblitera-

[1] Lectures in Pathology.

tion of the sac was often enough a postmortem discovery for which no symptoms during life had prepared us, and Osler confesses that it is more often discovered in the autopsy room than in the ward. In some cases this may be accounted for by lack of watchfulness on the part of the clinician, by the failure to correctly interpret the relationship of various physical signs, a too common fault, or the lack of attention to the heart when pericarditis comes late in a progressively fatal case.

ETIOLOGY. The principal change in recent years in the etiology of pericarditis is to regard all cases as of infectious origin. Osler,[2] in 1902, said that so-called idiopathic pericarditis was rare, but cases could occur, chiefly in children, in whom there was no evidence of rheumatism or any local or general disease. McFarland[3] says that there is always a focus of infection, but it is sometimes undiscoverable, and he quotes Bauer, of Munich, who states that out of 3000 autopsies occurring yearly in his city he sees only two or three cases of so-called idiopathic pericarditis. Demiéville[4] found but one case in the literature of twenty years, Virchow's case of hemorrhagic tuberculous pericarditis. Mallory[5] says that a variety of infectious agents may cause acute inflammation of the pericardial cavity. The most common are the Diplococcus pneumoniæ, the Streptococcus pyogenes, the Staphylococcus aureus, and the tubercle bacillus. They probably never lodge here primarily, but come from lesions elsewhere—thus by direct extension or through lymphatics from the lung, mediastinum, or heart, or by the blood stream from any part of the body.

Preble[6] in his series found pericarditis as a complication in pneumonia, 34 per cent; rheumatism, 28.36 per cent.; chronic diffuse nephritis, 11.20 per cent.; tuberculosis, 10 per cent.; sepsis, 4.7 per cent.; aneurysm, 2.6 per cent.; typhoid, 1.7 per cent.

Sears[7] in his report found rheumatism associated in 51 of his 100 cases, and a previous history with 40 others. Pneumonia or infection with the pneumococcus was next with 18. In 7 cases chronic nephritis was the chief cause and in 5 pleurisy—chronic rheumatism and gonorrhea are both credited with 2 cases while 1 occurred in each of the following diseases: empyema, tonsillar abscess, acute nephritis, hepatic cirrhosis, and chronic fibrous pneumonia, which was apparently not tuberculous. There were 9 cases of idiopathic pericarditis, but when we consider that some dated back as far as 1882 it is not remarkable that 9 out of 100 should be so classed.

Lamar and Meltzer[8] produced pneumonia in forty-two dogs by

[2] Practice of Medicine, 1902.
[3] Jour. Am. Med. Assn., December 7, 1901.
[4] Rev. méd. de la Suisse romande, Geneva, 1911, xxxi, 449.
[5] Principles of Pathological Histology, 1914.
[6] Jour. Am. Med. Assn., December 7, 1901.
[7] Med. and Surg. Reports, Boston City Hospital, 1897.
[8] Jour. Exper. Med., 1912, xv, No. 2.

means of intrabronchial insufflation, using pure cultures of pneumococci in suspension. They produced a lobar pneumonia in each instance. The findings in the fatal cases were similar to those in man: empyema, pericarditis, septicemia. In the fatal cases the pleural cavities contained a large quantity of bloody fibrinopurulent exudate, the pericardium was inflamed, and a septicemia existed. The exudate from the lung, pleural and pericardial cavities contained as well as the blood, large numbers of capsulated pneumococci in pairs and short chains. In their non-fatal cases it does not appear that the pericardium was involved, although much the same lung area was consolidated, but not as extensively. It would appear, therefore, that these results indicate that the pericarditis was the result of a profound septicemia.

The writer, working in the laboratory of Professor H. T. Karsner, produced pneumonia in ten dogs by the intrabronchial insufflation of pneumococcus cultures. Every effort was made to prove the organism before and after injection. One dog died on the tenth day, showing inflammatory changes in the lungs, kidney, spleen, liver, and heart. In the other nine dogs the pneumonia ran a mild course, the kidneys and liver showed moderate changes, but the hearts were normal. As far as the heart was concerned in the writer's fatal and non-fatal cases the involvement occurred only in the presence of a profound septicemia.

Torrey and Rahe[9] in their studies in canine distemper occurring in a series of ninety dogs found areas of consolidation in over 90 per cent. of lungs, while pericarditis was observed in only 5 cases, all terminating fatally. In 2 the condition was associated with pleuritis, and in 1 instance, Streptococcus pyogenes, while in the others it was sterile. The Bacillus bronchisepticus readily produces pneumonia but not pericarditis.

E. C. Rosenow[10] shows in his animal experiments with streptococci from rheumatic fever the greatest number of cases of pericardial infection corresponding with the observations of various clinicians. The interesting clinical point is that when he allows a reduction in virulence by keeping the organism in culture media there is a marked falling off in the effect of the organism on joints, myocardium and endocardium, while the pericardium does not become involved; but when the virulence is increased by animal passage the effect is less upon the joints and more on the endocardium and pericardium, especially the pericardium.

Robin and Fiessinger[11] state that gonococcus infections seldom attack the pericardium. These authors quote d'Argis, who, in 1895, collected 13 cases, and they report one case of their own. Their patient was a male, aged twenty-five years, with urethral discharge

[9] Jour. Exper. Med., January, 1913.
[10] Jour. Am. Med. Assn., November 13, 1915.
[11] Soc. méd. des. hôp. de Paris, 1912, xxxiv, 802.

and acute rheumatism complicated by signs of pericardial fluid. They aspirated at Marfan's point (base of xiphoid) and removed a fibrinous serum which did not show gonococci. The patient was treated with injections of Wright's vaccines, and made a good recovery.

The writer has not seen a case of gonorrheal pericarditis, and in the 78 Boston City Hospital autopsy cases in the last ten years there is not one recorded.

Triboulet and Harvier[12] report a case of severe typhoid in a boy, aged eleven and a half years, death being due to perforation. Acute pericarditis was unsuspected during life, but at the autopsy 90 grams of fluid were found in the pericardial sac. Cultures from the fluid gave a growth of the typhoid bacillus.

A. Pissary[13] mentions pneumonia, bronchopneumonia (especially in children), gonorrhea, scarlatina, typhoid, tuberculosis, puerperal septicemia, streptococcus infections, but places rheumatism first in etiology.

Mobécourt[14] gives an interesting case of streptococcus pericarditis in an infant; infection through the umbilical vessels at the time of birth produced a purulent peritonitis, with secondary purulent pericarditis. The purulent form, this writer thinks, is frequent in infancy because there is small defence against pyogenic organisms. This has not been the experience of Barkan and Lucas,[15] but all these authors (Bovaird[16]) agree that in small children the pneumococcus is the most frequent cause. Their articles contain exhaustive statistics on the etiology in infants and small children.

Marfan[17] says that pneumonia in children under six years and in older children and adults, acute articular rheumatism are the primary causes of pericarditis.

Of the 78 proved cases in our series, in 58 some form of acute infection was recognized before death. Pneumonia was diagnosed 28 times and acute pneumonia or pleuritis was found 49 times at autopsy. Of the 28 diagnoses of acute pneumonia, acute pericarditis was discovered before death in only 4 instances. It occurred in diphtheria 4 times, in chronic interstitial nephritis, 3; appendix abscess, 1; cerebrospinal meningitis, 2; scarlet fever, 5; general peritonitis, 3; chronic cardiac, 4; malignant endocarditis, 3; gallstones, 1; thoracic aneurysm, 3; erysipelas, arteriosclerosis, ulcerative colitis (TB) and gangrene once each. The clinical diagnosis of acute arthritis does not appear in the series, and while we have all seen acute pericarditis accompany this condition many times the mortality must be low as compared with pneumonia.

[12] Bull. Soc. méd. de hôp. de Paris, 1911.
[13] Clinique, Paris, 1913, viii, 756-759.
[14] Jour. de méd. de Paris, 1913, xxv, 383-388.
[15] Boston Med. and Surg. Jour., 1912, clxvi, 444-448.
[16] Med. and Surg. Report, Presbyterian Hospital, New York, 1912.
[17] Semaine méd., Paris, 1913, xxxiii.

The question of extension of the inflammation from adjacent organs, especially in pleuritis and pneumonia, seems still in doubt. Osler[18] believed that it could occur by extension as a serious complication in pneumonia, and he reported, in 1902, that it was present in 5 cases of 100 postmortems he made at the Montreal General Hospital. He states that in simple pleurisy it is rare.

G. A. Gibson,[19] in 1898, said that it often had its origin by extension in pleurisy and pleuropneumonia as well as in endocarditis and myocarditis, but he furthermore states that it must not be forgotten that in pneumonia the source of the pericardial infection may be through organisms conveyed by the blood, and pericarditis from pneumococci has been seen without pneumonia. Septic diseases of the mediastinal glands, mediastinal tumor (Bergmann[20]), as well as destructive processes in the lungs, may produce pericarditis by extension, while acute affections of the bones—the sternum and ribs in front and the vertebræ behind have been seen to give rise to it. In one of our cases it was due to sarcoma of the mediastinum, but it was not classed as acute pericarditis.

Preble[21] lays down rather definite statistics showing the ratio of pericarditis to the area of lung involved in pneumonia. He says that with unilobar pneumonia the chances of a pericarditis are 1 in 40; with a bilobar or trilobar, 1 in 10; with a quadrilobar, 1 in 5; with a pneumonia of the entire left lobe, 1 in 8. He feels that the danger is somewhat greater with a left than with a right-sided pneumonia. The writer has not found another author who has laid down such definite lines.

Mallory in his book states that pericarditis may occur by direct extension, and Professor Councilman makes a similar assertion in his lectures.

Sears[22] in a study of 100 cases found 34, with evidence of some previous valvular or myocardial affection, and while he says it may be a coincidence, the number is so large that it is more probable that the preëxistent lesions acted as a determining cause. Many of his cases were complicated by, or secondary to, pneumonia, pleurisy, or both, either by direct extension or from a simultaneous infection.

Brooks and Lippencott[23] found in their 150 cases of pericarditis, pleural lesions present in 136. In well over half of these cases it appears that the pleural change is the more anterior, and that the pericardial alteration occurred later, though still back of this relationship is the general disease or condition in the course of which the two membranous inflammations developed. They found a direct relationship between pericardial and pleural lesions best

[18] Practice of Medicine, 1902.
[19] Diseases of the Heart, 1898.
[20] Charité-Ann., Berlin, 1909, xxxiii, 92-101.
[21] Loc. cit.
[22] Loc. cit.
[23] Am. Journ. Med. Sc., December, 1909.

shown in the acute serofibrinous cases, in which out of a total of 67 instances, 60 showed pleural changes. In 36 of these the lesions were identical. These authors were unable to trace any direct relationship between pericardial and endocardial disease. For example, in their serofibrinous cases, 46 of which were caused by bacteremia of some variety, only 10 showed acute endocardial changes; 7 of these were unquestionably rheumatic and bore no other relation to the pericarditis than that both had probably a common etiology.

Sinnhuber,[24] believes that acute articular rheumatism is the foremost cause of pericarditis, then pneumonia, tuberculosis, scarlet fever, influenza, typhus abdominalis, recurring fever, cerebrospinal meningitis, malaria, erysipelas, osteomyelitis, pyemia and sepsis, gonorrhea, scurvy, leukemia, and certain forms of cirrhosis of the liver. He also states that it can arise from diseases of the neighboring organs, endocarditis, myocarditis, diseases of the pleura, especially on the left side, perforation of the esophagus from a foreign body, caries of the sternum and ribs, and as a sequel of aneurysm.

In our series cultures were not always made, but in the 78 cases the streptococcus was recovered 19 times, the pneumococcus 17 times and staphylococcus 8. In one of the cases of cerebrospinal meningitis the meningococcus was recovered from the brain and pericardium, and in the other case the heart's blood showed streptococci, and the pericardium, staphylococci.

SYMPTOMS. The symptoms of pericarditis are so varied and the onset so insidious in some cases that Osler and almost every other author has been led to say that many cases run their entire course without recognition. When it comes on very gradually, or is the only manifestation of an obscure focus, or is well established when the patient comes under observation, detection is sometimes most difficult if not impossible. The importance then of a thorough physical examination of the heart and lungs at the first visit, with subsequent daily examination augmented by careful notes and charts, is obvious. It is well to mark out the percussion lines on the chest and to test them daily for any change. The intensity of all the heart sounds, their relation to one another, and the character of murmurs should be carefully noted.

Pain is an important subjective sign and one which a good many practitioners expect to find. Pleurisy is almost immediately painful, and so it is thought that pericarditis will be; but one should not expect to have the patient complain of pain as he does in pleuritis.

A. McPhedran[25] says that pain is generally present, and quotes Sibson, who found it in 70 per cent. of his cases. L. V. Schrötter[26]

[24] Die Erkrankungen des Herzbeutels und ihre Behandlung, Berlin, 1911.
[25] Osler's Modern Medicine, 1908.
[26] Nothnagel's Practice.

states that the greater number of cases occur without pain. James Mackenzie[27] calls attention to the absence of pain in a good many cases. Poynton[28] says it may occur with little discomfort and no pain. Pain was present in 65 cases in the series of 100 reported by Sears, and when absent its place was taken by a sense of oppression in the chest.

Babcock[29] found pain in the majority of his cases, or if not, a sense of distress.

It has seemed to the writer that a good many patients with acute disease of the heart, while they do not have actual pain in the heart, still when one asks if ice over the precordia makes them feel more comfortable, they generally say that they prefer to have the ice kept on.

Blumer[30] states that pericarditis is often almost without pain.

Billings[31] does not mention pain in his text, and in only one case history.

In the protocols of the 78 cases in our series, pain was not a striking feature, and was observed 26 times—in 17 of the 26 cases there was either acute pneumonia or disease of the pleura, in 2 others chronic disease of the myocardium, and in 1 aneurysm of the arch of the aorta, leaving but 6 cases out of the 26 with pain directly over the precordia.

In the 2 cases of purulent pericarditis with operation (one recovered, the other died without autopsy) pain was not observed at any time.

It has been the writer's experience repeatedly in cases of slight dry and serous pericarditis lasting a few days to note the absence of pain, although in this study no conclusions from figures can be given.

It must be remembered that many of the patients in our series were seriously ill at the time of entrance to the hospital, and their general distress was so great that they may not have noticed pericardial pain.

The cause of pain is difficult to determine. The pericardium, like other membranes, the peritoneum and pleura, are insensitive when normal, but if inflamed, pain is produced. Special work seems necessary on this subject.

Poirier[32] says it is very probable that the left pneumogastric and the nerves of the cardiac plexus give off also several nerve fibers to the fibrous layer.

The skin over the precordium is sometimes tender because of the connection between the upper intercostal nerves and the ganglia and nerves of the cardiac plexus (Piersol[33]).

[27] Diseases of the Heart, 1908. [28] Pediatrics, New York, 1910, xxii, 353-363.
[29] Diseases of the Heart and Arterial System, 1905.
[30] Jour. Am. Med. Assn., July 18, 1914.
[31] Ibid., December 7, 1901.
[32] Anatomy.
[33] Anatomy.

Krehl[34] quotes Goldscheider,[35] who denies the existence of special nerves of pain. He continues with the statement that every sensation produces at the same time a more or less definite impression of the place whence the sensation has come. In the case of the eyes and skin the localization is very accurate; in the case of the mucous membranes near the outside of the body it is somewhat less accurate; in the case of the mucous membranes and the organs within the body it is inaccurate and entirely unreliable. Howell[36] also makes this last statement.

Allbutt[37] states that there are cases in which the pain is like that of angina pectoris, and refers to several observers, Sibson, Byron Bramwell and others.

In studying the subjective symptoms of pain and a feeling of distress in the precordia or chest in pericarditis, it seems to me that it makes a great difference whether one builds his statistics of pain on clinical or autopsy observations. When one considers that only about 50 per cent. of the cases are recognized, and of that number about one-half have precordial pain, it makes the pain a small factor.

Pericardial Friction. In a pericarditis which remains dry or rather has but a moderate increase in fluid the sole physical sign is the to-and-fro friction rub. It is generally heard, if only for a few hours, in almost every case of plastic pericarditis.

Billings[38] says it is present in every case at some time in its course, and by systematic examination it will be found.

McPhedran[39] thinks there are many cases in which it is not found, "but once distinctly heard it can scarcely be confounded with anything else." The writer feels hardly able to make such a sweeping statement. Meeting a considerable number of students of all ages every year in the Graduate School of Medicine of Harvard University, it is uncommon to have a student recognize a pericardial friction rub. Three years ago a very able practitioner from Vermont told me that he had been in practice thirty years and had never heard a pericardial rub until he came to our clinic. This is not surprising when one considers that acute pericarditis is not such a very common complication, having been diagnosed clinically but 100 times in 34,467 medical patients treated in the wards of the Boston City Hospital during the last ten years by physicians giving a good deal of time and thought to diseases of the chest. Acute pericarditis has been found 78 times in 1553 autopsies during the same period.

Many writers speak of the pericardial changes beginning at the point where the membrane is in contact with the great vessels,

[34] Clinical Pathology, 1909.
[35] Ueber den Schmerz, Berlin, 1894.
[36] Text-book of Physiology, 1913.
[37] Diseases of the Arteries, 1915.
[38] Loc. cit.
[39] Loc. cit.

because the infecting agent is carried to the sac by the blood, and at that point the blood supply is richer.

Rosenow[40] found in the pericarditis of animals that hemorrhages usually surrounded a relatively large artery. In some of the sections the artery was the seat of thrombosis, or accumulation of leukocytes along the intima in the area of the hemorrhages. Some of the leukocytes contained organisms, and in one instance typical organisms were found directly in the wall of an artery which showed mural implantation of leukocytes adjacent to an area of hemorrhage in which exactly similar organisms were found in small numbers.

Mallory[41] says that it may occur anywhere in the pericardial sac. In one case of the series of 78 autopsies of acute pericarditis the inflammation was found about the great vessels, but more often it involved the entire sac. In animals the focus can be more readily studied while the human subject often does not die until the entire sac is involved.

In the experiments of Walker and Christian[42] in the production of pericarditis by the injection of spartein sulphate and adrenalin chloride, the inflammation was usually adjacent to the left ventricle.

The chief difficulty seems to be in distinguishing the rub from a valve murmur. The rub even when faint will generally have a jerky, grinding, or leathery quality. Because of the more active movements of systole it may seem to be wholly a systolic sound, but by listening carefully a sound of similar quality, sometimes very faint, will be heard in diastole. The rub may be mistaken for a valve murmur even by the most experienced. Three years ago, when looking for a case of aortic stenosis to show the students, I was told by the house physician of a colleague, distinguished as a specialist in circulatory diseases, that they had a case in Ward M. The patient, a man, aged twenty-seven years, was convalescent from an acute multiple arthritis. He looked well and said that he was absolutely free of his discomforts. On listening to his heart he was found to have over the second interspace to the right of the sternum a rough, grating murmur, systolic in time. On listening carefully, however, a similar murmur, very faint, was heard in diastole. There were absolutely no cardiac or circulatory signs indicating aortic stenosis or regurgitation. In the absence of these confirmatory signs of valvular disease, outside of the precordia and taking into account the history of recent rheumatism, it seemed more probable that he had a pericardial friction rub. Two days later the rough sound, with its fainter diastolic, was heard over the third interspace to the left of the sternum, the following day over the fourth, and by the fifth day it was barely audible just above the cardiac impulse, and then disappeared. Within two months a consultation was held at the hospital to decide whether a patient

[40] Jour. Infect. Dis., May 16, 1915.
[41] Loc. cit.
[42] Tr. Assn. Am. Phys., 1910.

had double aortic disease or pericarditis. The same considerations decided in favor of pericarditis.

The rub often moves from day to day, and is sometimes made more intense by pressure with the stethoscope. When the rub disappears it may mean that the plastic pericarditis is quieting down or fluid is accumulating.

Fluid. In many cases, the majority in fact, a little increase in fluid occurs and then quiets down. It is not recognized clinically, as it gives no physical signs because the sac is distensible and the added pressure causes no embarrassment to the heart or circulation.

Poynton[43] says that paracentesis is rarely needed in childhood, thereby indicating the frequency of spontaneous absorption.

The cardinal points which have always been taught are increase in the cardiohepatic angle, increasing faintness (distant) of the cardiac sounds, the disappearance of the cardiac impulse, and the pulsus paradoxus.

Percussion should be performed daily or oftener marking out carefully the cardiac borders. The angle of cardiohepatic dulness will be found to increase with increasing fluid. James Mackenzie[44] says that the dulness will also reach up to and above the second rib and the area mapped out will have a somewhat pear-shape appearance. This shape has been seen several times by the writer, and in a roentgenogram the enlarged area looks like a paper bag filled with water and set down upon a table. In comparing these roentgenograms with those from dilated hearts the line in the latter is almost always a curve inward as it approaches the liver. If the increase is due to enlarged liver it can generally be distinguished by a study of other pericardial signs of effusion, such as a faintness of heart sounds and disappearance of the cardiac impulse.

Emphysema may mask the heart sounds so that there may be poor sounds with a good pulse, as is often the case in pericardial effusion. When there is a left pleural effusion the heart may be pushed to the right and the border between the pleura and the pericardium will be lost. Aspiration of the pleural fluid should reduce the cardiohepatic angle, and if it does not, after the heart has had time to return to its normal position, pericardial fluid should be suspected (Cabot[45]).

Shifting dulness has not been helpful to the writer. The fluid is generally under such tension between the myocardium and the fibrous parietal pericardium that it is not clear how it could shift. When the fluid is posterior the heart may be pushed forward and there may be no diminution in the cardiac impulse. In a case seen in consultation the increasing cardiohepatic angle and signs of distress raised the question of fluid. A roentgenogram showed the characteristic pear-shaped enlargement; still, we hesitated to aspirate

[43] Loc. cit.
[44] Diseases of the Heart, 1914.
[45] Physical Diagnosis.

because of the very distinct cardiac impulse. A second roentgenogram was taken, showing an increase in size, and upon aspiration about 150 c.c. of bloody serum were removed, with considerable relief to the patient. In these cases almost all authors, Billings,[46] Babcock,[47] J. Mackenzie,[48] Cabot,[49] Musser,[50] Parkinson,[51] call attention to the small area of dulness in the left back just inside the angle of the scapula, described by Ewart, and produced by mechanical atelectasis of the compressed lung.

Moog[52] adds one case to a report of four others of pericardial effusion punctured in the rear. He tapped the pericardium through the fifth interspace in front and evacuated 130 c.c. of hemorrhagic fluid; two days later he evacuated 90 c.c. in the same way. The symptoms from compression of the lung soon returned, but two punctures at different points brought no fluid. Then he introduced the needle in the rear in the eighth space and obtained 600 c.c. of the effusion, the same amount the next day; and five days later 400 c.c. Air got in during the later puncture, and pneumothorax developed with some effusion, but this gradually disappeared with all the other symptoms. The patient made a good recovery.

Pauly[53] calls attention to the signs in the back.

Curschmann[54] reports 3 cases of successful paracentisis in the back at the eighth interspace.

Mackenzie[55] reports having tapped a purulent pericardial effusion from the back in mistake for an empyema, his mistake arising from not having ascertained the position of the heart's movements. He says that had the case been one of pleural effusion he would have found the heart beating to the right of the sternum, but the whole left chest was dull, so that the idea of it being a pericardial effusion did not occur to him. One of my hospital colleagues told me that he made a similar mistake two or three years ago.

R. C. Larrabee and the writer tapped a boy, aged ten years, just in front of the left midaxillary line, removing several ounces of bloody fluid. The almost immediate cessation of cardiac distress convinced us that we had put the needle into the pericardial instead of the pleural cavity.

The pulsus paradoxus, which is small and feeble during inspiration, was formerly considered as important, but it occurs in other conditions and even in the normal subject.

Musser,[56] Cabot,[57] West,[58] and many others mention it, but do not regard it as important. In one case in the writer's series it was noted.

[46] Loc. cit.
[47] Loc. cit.
[48] Medical Diagnisis, 1913.
[49] Therap. Monat., Berlin, June, 1914.
[50] Therap. Monat., Berlin, 1912, xxvi.
[51] Loc. cit.
[52] Lancet, London, 1910, i, 560-564.
[53] Loc. cit.
[54] Loc. cit.
[55] Clin. Jour., London, 1912, xl, 45.
[56] Rev. de méd., Paris, 1911, xxxi.
[57] Loc. cit.
[58] Loc. cit.

Taussig[59] says that ordinarily the dicrotic wave in the radial sphygmogram does not vary in size with the phase of respiration. In pericarditis, however, it becomes more or less exaggerated, sometimes greatly so, during inspiration. He studied 5 cases, making this observation in all.

The writer has not had experience with the following signs:

Wynter[60] believes that absence of the abdominal respiratory movement is an indication of pericarditis. He reports 2 cases in which the remarkable stillness of the abdomen led to a diagnosis of acute abdominal lesions, pericarditis being discovered later. It is a valuable sign, he thinks, as it may precede and outlast other indications. The inflammation of the pericardium may cause reflex inhibition of abdominal movement some days before the appearance of the ordinary clinical signs. It is more apparent with a fibrinous exudate. He reports 10 other cases in which he found this sign.

When we remember that the base of the pericardial sac rests upon the diaphragm it is quite as possible to give abdominal symptoms as in the well-known instances of inflammations of the pleura and lung.

In the only cases in our series in which abdominal pain was noted there was definite disease of the abdomen.

Cobb[61] has reported a case of acute serohemorrhagic pericarditis in a boy, aged eleven years. He had never been sick until ten days before Cobb saw him, when he had an acute coryza, with the exception that he complained of a little pain at the apex of the left lung on deep inspiration. His physician found nothing but a few slight friction rubs, which disappeared within twenty-four hours. The temperature did not go above 100.5° F., and became normal at the end of the third day. Three days after apparent recovery and five days before Cobb saw him he ate heartily of apples and ice-cream, was seized with a sudden attack of abdominal pain, which made him cry out and writhe on the floor. He vomited several times in this attack; the temperature was 100° F., but nothing could be found on physical examination. He recovered in a few hours and was apparently as well as ever. Three days later he had some nausea and vomiting, and complained of abdominal pain. This was followed by a day of comfort, when he again had nausea, vomiting, epigastric pain, and pain in the right hypochondrium well down in the flank. He was apparently very sick, with a rapid and irregular pulse. In the epigastrium and right and left hypochondrium there was exquisite tenderness. After careful examination of the chest, nothing abnormal was found except the respiration in the left back was somewhat shallow, apparently due to the tenseness in which

[59] Jour. Am. Med. Assn., June 13, 1914.
[60] Clin. Jour., London, 1911–12, xxxix, 218.
[61] Ann. Surg., October, 1912, lxi.

the abdominal muscles were held because of the pain. The heart was normal in size and situation. Upon opening the abdomen in the median line nothing was found except a very large and congested liver. At the autopsy the pericardium was found to be 5 mm. thick and contained a considerable amount of bloody fluid with soft fibrin, free and attached. Cultures were not reported.

The writer thinks that the shallow breathing in the left back may have been due to the pericardial pressure—Ewart's sign; the congested liver, to pressure about the great veins within the sac. The heart valves were normal.

I was once asked to see a physician's wife who had been taken to a private hospital with a diagnosis of appendicitis. Certain features in the case prompted the surgeon to ask for a medical consultation. The history was that she became easily tired and a little short of breath on exertion. She had had three attacks of abdominal pain, which seemed to bear a relation to her menstruation. On physical examination a marked mitral stenosis was found and an enlarged and tender liver. By resting the heart the liver reduced in size and the attacks of pain ceased.

Calvert[62] believes that in pericarditis with effusion the right lobe of the liver is low, varying in its position from the 5th space to the 6th rib; in dilatation of the heart the right lobe of the liver is high from the 4th to the 5th rib. He reports 2 cases of pericarditis to confirm this.

The writer has not met with this sign nor has he found mention of it elsewhere, but it seems extremely doubtful if it would give much help. Turrettini[63] studied a sign noted by Josserand,[64] and Mouriquand and Roubier[65] of impending pericarditis or endocarditis. Turrettini observed this sign, "Éclat clangoreaux disastolique au foyer pulmonaire" in 2 cases, a sudden and violent clanging diastolic sound in the pulmonary area. The pulmonary second sound was very pronounced and the vibration could be felt by the hand. The second aortic sound was perceptible, but less marked, a friction rub developed soon after. His cases are not very helpful, as one patient had chronic nephritis with hypertrophy and dilatation and the fibrinous pericarditis developed slowly while the patient was under treatment, and the other was similar without autopsy. In two other cases of pericarditis from the spread of a pleural process this peculiar clang was never heard. He believes the sound to be important because it enables one to recognize incipient pericarditis, and by instituting treatment in time to possibly ward it off. The writer does not find in the literature any other observers who have made use of this sign. In chronic nephritis the diastolic

[62] Arch. Int. Med., 1909, iii, 92; Jour. Am. Med. Assn., August 27, 1910.
[63] Rev. méd. de la Suisse romande, Geneva, 1913, xxxiii.
[64] Lyon méd., November 17, 1907.
[65] Ibid., September 1, 1907.

second sound is often sharp and ringing, until the systolic blood-pressure falls and cardiac compensation fails.

The knee-chest position has been considered by Rieux[66] to be of diagnostic value. The writer has never seen a case of pericarditis with this sign. In diseases of the chest patients assume the most comfortable attitude for breathing.

The diagnosis in dry pericarditis rests solely upon the to-and-fro friction rub. The fluid which usually follows may be so slightly in excess of the normal as to escape detection and with the disappearance of the rub nothing further may be found, or there may be a gradual or rapid change in the area, the obliteration of the cardiohepatic angle, dulness extending to and above the second rib; the disappearance of the cardiac impulse; the faintness of the cardiac sounds; increasing cardiac distress sometimes out of proportion to the cardiac findings; cyanosis and signs of embarrassed circulation. In a man, aged thirty-two years, who had been ill two weeks with pneumonia, was getting better and then began to grow worse, the right border of the heart was 7.5 cm. from midsternum, the left border 14 cm. The apex beat was indistinctly felt in the 5th space within the nipple line. The cardiohepatic angle was distinctly obtuse. The striking point was the difficulty of hearing anything in the way of heart sounds. The abdomen was practically negative, and there was no sign (Ewart) in the back. Dr. G. G. Sears, who saw this patient shortly after he entered the hospital, immediately made a diagnosis of pericarditis with effusion, based on the rapidity of respiration, hoarseness of voice, size of cardiac dulness, and lack of heart sound with a fairly good pulse. The roentgen-ray confirmed the diagnosis. A needle inserted in the fifth space at the left outer border of cardiac dulness gave 20 c.c. of seropurulent fluid. At operation about 2 quarts of pus were removed. The patient seemed relieved, but died a few hours later.

Sears[67] has said that unresolved pneumonia is a pathological myth. If the temperature keeps up and the signs persist after due course of time, something is amiss. If we can exclude complications outside the lungs as a cause of fever, *e. g.*, otitis media, pericarditis or endocarditis, one arrives at a diagnosis by exclusion of either pulmonary abscess or empyema, and of these the latter is vastly more common. These words might also be used in discussing pericarditis; sometimes the pleural cavity is aspirated first in doubtful cases (Ortner[68]); Harrigan[69] thus excluding paracentesis of the pericardium or proving its necessity.

We will speak of paracentesis as a diagnostic as well as a therapeutic procedure. It is necessary in cases progressing unfavorably

[66] Paris méd., 1912-13, xi, 185-191.
[67] Boston Med. and Surg. Jour., November 25, 1915.
[68] Deutsch. med. Wchnschr., Leipzig., 1910, xxxvi.
[69] Ann. Surg., lvii, 1913.

to determine the plan of treatment. The point of puncture is decided by the physical signs and the roentgen-ray.

Pendlebury[70] says the spot chosen for introduction of the aspirating needle, of not less than one-tenth in diameter, is in the 5th left interspace, one inch from the margin of the sternum. By choosing this spot injury to the pleura and danger of wounding the internal mammary artery will be avoided. The most careful aseptic precautions must be observed, and he advises incising the skin before putting in the needle. He urges careful attention to the first few drops of fluid to avoid injury to the heart. These directions are also given by Friedrich Pels-Leusden,[71] who adds that we may in the above method occasionally prick the heart, and furthermore says that we may go in the mammary line in the 5th space in which the mammary artery and the heart are not imperilled, but where we must necessarily go through the pleura. Curschmann advises puncture always far outside at the limit of dulness and to go without fear through the pleural cavity, even if the latter is matted down by adhesions, so as to be sure not to injure the heart.

Ogle[72] advises introduction of the needle slowly and steadily, and as soon as it is through the skin the aspirator should be connected and the glass index watched for the first indication of fluid.

The writer has had his chief experience with the above method. Blechmann[73] in an exhaustive article discusses the various methods of exploratory puncture. The one already described by Pendlebury (Dieulafoy[74]), the left parasternal (Delorme and Mignon[75]), the right parasternal (Rotch[76]), and Marfan's[77] epigastric xiphoid route.

As Curschmann says, no definite rule can be laid down in any case. We must be guided by the area of dulness, the findings by exploratory puncture and the roentgen-ray.

In our other case of purulent pericarditis following pneumonia the right border of cardiac dulness extended until within 2 cm. of the right nipple, and exploratory puncture was made in the 4th space to the right of the sternal margin and the operation followed the same line, the 4th and 5th costal cartilages being resected. This patient made a good recovery and the discharge ceased in three weeks.

The chief worry of the novice in exploratory puncture is the fear of wounding the heart. Sears[78] says: "That the danger arising from wounding the heart is theoretical rather than real received

[70] Latham and English, System of Treatment, 1915.
[71] Handbook of Surgical Operations.
[72] Latham and English, 1915.
[73] Semaine méd., 1913, xxxiii.
[74] Traité de l'aspir. des liquides morbides, Paris, 1873.
[75] Rev. de Chir., 1895, 797–787.
[76] Boston Med. and Surg. Jour., 1878, xcix.
[77] Semaine méd., Paris, 1913, xxxiii, 469–476.
[78] Boston City Hospital, Med. and Surg. Report.

another illustration in one of these cases when the needle thrust into the 6th interspace just to the right of the sternum entered a solid body which pulsated and caused it to describe an arc of one and a quarter inches with each beat. No harm results."

Ewart considers the removal of blood sometimes most beneficial, and Sharkey[79] reported a case in which the blood came through the trocar in jets. It did the boy a great amount of good.

Byron Bramwell[80] reports a case of continued hemorrhage into the pericardium from puncture of the ventricle.

In rabbits, blood is removed from the ventricle for therapeutic purposes without doing the rabbit any harm.

Cushing and Branch[81] in their report on work done in the production of chronic valvular lesions in dogs found that in spite of the most rigid asepsis neither the pericardium nor the pleura withstand operative measures with anything like the resistance which the peritoneum exhibits in the face of similar manipulations. A considerable number of the animals which recovered and were subsequently sacrificed showed evidences of a slight degree of infection. They add, however, that no one who has not had experience with operations on animals can have any idea of the amount of handling the heart will stand without injury.

In Harrigan's[82] case of temporary arrest of the heart beats following incision of the pericardium for suppurative pericarditis, the heart was seen lying still, but it began to beat again as soon as gauze was introduced into the sac. This was a feeble child much exhausted by a long-standing infection, but recovered from the immediate effects of the operation.

Sears calls attention to the fact that the heart is more apt to show signs of embarrassment with arrhythmia and enfeeblement of the pulse when the effusion begins to form, later when the sac begins to distend the circulatory signs are less urgent. In accord with this are the findings of Heitler,[83] who electrically and mechanically stimulated the visceral pericardium, thereby considerably disturbing the cardiac rhythm even after section of the cardiac nerves. He found the left ventricle excitable, especially over the upper third; the right ventricle is not markedly excitable except close to the sulcus, while the apex is variable.

Mackenzie[84] has never found any serious embarrassment from extensive pericardial effusion. The pericardium normally is an inelastic bag, but with inflammation it becomes distensible.

Chatin[85] found that the amount of fluid which could be forced into a normal pericardium was 700 c.c., but in inflammation very

[79] Lancet, December 6, 1902.
[80] Clinical Studies.
[81] Jour. Med. Research, February, 1908.
[82] Loc. cit.
[83] Wiener klin. Wchnschr., 1898, No. 3.
[84] Loc. cit.
[85] Rev. de méd., June 10, 1900.

large amounts may accumulate. Kay[86] reports a case in which at autopsy 3500 to 4000 c.c. were found.

When the fluid is in large amount the pressure symptoms on the auricles and great veins may become marked, and death may result. Another danger (M'Phedran[87]) is the interference by pressure with the coronary blood supply, this favoring degenerative changes in the structure of the heart.

TREATMENT. The question of injection into the pericardial sac after removal of the fluid is unsettled. Few observers can see any advantage, and almost all regard attempts at local medication to be harmful. Washing out the sac even with normal salt solution is without supporters. The object of any form of local treatment is to prevent adherent pericardium.

Wenckeback[88] obtained good results by introduction of air. He removed the fluid and put in one-half the volume of air. He suggests using nitrogen or oxygen.

Wilbur[89] quotes McPhedran, who advises in pyopericardium the injection of a 2 per cent. liquor formaldehyde in glycerin, as is sometimes used in empyema.

Rehn[90] injected dogs' hearts with tincture of iodin and with aleuronat, produced synechia, and then checked adhesions by injections of sesame oil. Wilbur suggests the use of some oil, such as liquid petroleum.

In pyopericardium, free incision with drainage is indicated as in empyema. Manual removal of fibrin is sometimes necessary.

Medical treatment is thoroughly discussed in several good text-books. Ice is of value and is used in every case in the Boston City Hospital whether there is pain or not. I always ask the patient if it gives him comfort, and he generally replies in the affirmative. Be sure that the ice-bag is not too heavy—it is sometimes an added burden to a laboring chest.

Rubino[91] found that he was able to produce pericarditis in animals by the intravenous injection of cultures of pyogenic cocci only when ice was simultaneously applied to the chest. On this ground, ice has been objected to as a therapeutic measure, but Rubino probably severely chilled the animals and lowered their resistance.

The treatment by drugs is nearly useless, but whatever is done is generally indicated by the infective process.

In rheumatism the salicylates are of undoubted value. It has been said that they sometimes depress the heart, but it is more probable that the depression comes from the toxemia.

[86] Penna. Med. Jour., 1911–12, xv.
[87] Ztschr. f. klin. Med., Berlin, 1910, lxxi.
[88] Jour. Am. Med. Assn., July 25, 1914.
[89] Loc. cit.
[90] Arch. f. klin. Surg., Berlin, 1913, cii.
[91] Arch. Ital. de Biol., 1892.

Pericarditis occurring before the crisis of pneumonia is apt to mean an overwhelming toxemia, and the prognosis is very grave. Later, after the patient has begun to improve, the effusion is often purulent, and the treatment has been discussed.

The diet should be light and easily assimilable, as in cardiac and acute infections.

Absolute rest in bed until the signs of pericarditis have ceased and the heart has fully regained its tone is of vital importance.

Conclusions. The conclusions from the literature and the series of cases are:

1. Acute pericarditis is a secondary affection. Several other lesions were found in every case in the 78 autopsies.

2. The extension from adjacent structures is probably uncommon, the vast majority of cases occurring from infection of the sac through the blood stream.

3. Pain in the precordial area is not as common as is supposed. A good many mild cases pass unnoticed because there is neither pain nor distress. In rheumatism with an acute heart the pain and arrhythmia may be noticeable at first, but later when all structures of the heart are involved the picture is one of general cardiac and circulatory embarrassment. Pain is so much more common in pleuritis, and pneumonia plays such a factor in pericarditis, that it is often difficult to separate the two.

4. The difficulty in diagnosis is illustrated by the fact that acute pericarditis was recognized clinically only 100 times in 34,467 cases and 12 times in 78 autopsies at the Boston City Hospital. Pneumonia is the chief agent in the production of purulent and of fatal pericarditis. Rheumatism may cause a greater number of cases of all types, but the figures are open to doubt, as they include all grades of severity, and many can be studied from their inception, while in a rapidly progressing infection like pneumonia the changes in the heart may be masked by adjacent physical signs and the cardiac and respiratory distress of the patient. Rheumatic pericarditis should always be thought of, especially in young adults, when the signs of cardiac failure are out of proportion to the other cardiac physical findings. Pneumococcus pericarditis or myocarditis, or both, should be considered, especially in young or middle-aged adults, when the heart shows failure of compensation before the crisis or after it when there is fever and delayed convalescence.

5. The prognosis in acute pericarditis following acute arthritis is generally favorable to life; the extent of damage to the subsequent function of the heart cannot be determined, of course, immediately. In the clinical protocols of this series, acute arthritis was not found once. Pneumococcus pericarditis is grave at any stage. Tuberculous pericarditis is not common, and is usually a late involvement in an advanced case.

6. In mild cases of fibrinous pericarditis or of moderate effusion,

especially when an accompaniment of acute arthritis, treatment of the infection and local applications to the precordia will often suffice. When the progress is unsatisfactory, the physical signs increasing, and the diagnosis doubtful, paracentesis is imperative.

7. Frequent examinations during the infection and for some time after are very important. A thorough search for and the eradication of foci should be conducted in every case.

The writer wishes to thank Dr. Sears, Dr. Councilman, and Dr. Mallory for many valuable suggestions, and the members of the Visiting Staff of the Boston City Hospital for the privilege of referring to the records of their cases.

A TREATMENT OF GASTRIC ULCER BASED UPON ESTABLISHED CLINICAL, HISTOPATHOLOGICAL AND PHYSIOLOGICAL FACTS.[1]

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Even a casual acquaintance with the literature of peptic ulcer demonstrates that treatment of the affection has been largely empirical. Various types of therapy appear to have obtained a vogue because their application either relieved symptoms or appeared to prevent a fatal issue. Success of a mode of treatment has been judged mainly from its effect upon the immediate condition and not with respect to the ultimate outcome of the affection. It is most unusual to observe statistics of hospitals or private practices that indicate the status of patients treated after intervals of five to twenty-five years. Hospital records generally state that a peptic ulcer patient has been discharged either "cured," "improved," "not improved," or has "died." With exception of the fatal cases, facts are usually not available regarding the future course of the ailment, inasmuch as these patients frequently seek the advice of another physician should their disability return after a long and expensive course of treatment at the hands of a former physician. It is also of interest to observe that of a half-dozen experts non-surgically treating peptic ulcer by radically different regimes the percentages of cure show a range of but five to ten points. We have also been impressed by the fact that at the most competent hands, certain ulcers resisted all types of treatment alike. It has seemed to us that unsatisfactory as the treatment of peptic ulcer might be,

[1] Read at the Annual Meeting of the American Gastro-enterological Association, Washington, D. C., May 8 and 9, 1916.